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The project began as an investigation into the possible interaction between acute cold-induced hyperventilation and the hyperventilation which occurs with isometric exercise. Concern was raised regarding the potentially dangerous consequences of combining these stresses. Based upon the interaction and questions raised by these initial experiments, this aspect of the study was enlarged to include the study of the effects of cold water immersion on the ventilatory response to rhythmic exercise and the effects of habituation on the respiratory responses to both type of exercise.

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In fact, it emerged that the respiratory responses to immersion in cold water were not additive to those found during isometric or rhythmic exercise. The protocols were arranged to study the question both during the peak hyperventilation soon after immersion in cold water and also 10 minutes after immersion when the ventilation had subsided to a steady-state level. Regardless of the different protocols, it turned out that the level of hyperventilation that occurs in isometric contractions fell approximately linearly with decreasing water temperature (35°C to 15°C) which was the converse of our hypothesis. Similar, though less dramatic effects were found when the subjects performed rhythmic exercise. Nevertheless the result is clear that there is an inhibition of the ventilatory drive when humans exercise in progressively colder water. That raises the question of the mechanisms involved. The most likely explanation is that there is an inhibition of the neural traffic from the exercising muscles or from "central command" originating from cutaneous thermal receptors. Such phenomena ae known for suppression of pain but the suppression of such a vital function as ventilation seems unusual but, at the same time, protective. Had the hyperventilation of isometric contractions and immersion in cold water been additive, the alkalosis would have increased to a serious level.

There was no change in the cardiovascular responses to isometric contractions in cold water when compared to immersion in thermo-neutral water at 35°C. That was not surprising because the duration of immersion was not long enough to reduce the core or muscle temperatures to create adverse conditions. One unexpected finding from these experiments was that the fall in the temperature of the forearm muscles was identical when the subjects were fully immersed (to the neck) in cold water or when they immersed only the forearm; we had expected a more rapid fall in muscle temperature during whole-body immersion as a protective measure in terms of thermoregulation responses.

In contrast, the cardiovascular responses to rhythmic exercise in cold water were quite different from those found either in air or during immersion in thermo-neutral water at 35°C. Not surprisingly, the energy cost of rhythmic exercise was greater in water than in air and progressively so as the water temperature decreased. But in cold water at 20°C and less, the cardiac output for any given level of oxygen uptake was less than in thermo-neutral water or in air. The reason for this unexpected relationship is not clear. The principal cause was the change in heart rate with relatively small contributions from alterations in stroke volume, as would be expected when the subjects were immersed to the neck. A major question is whether there is a cold-induced restriction on cardiac output and if so, what the mechanism might be. In addition, the arterial blood pressure both at rest and during rhythmic exercise was higher during immersion in water at 20°C than in any other experimental condition. Again, this points to a stimulus from the cutaneous thermal receptors. The subjective sensations in water at that temperature were the most uncomfortable; in water at 15°C, the skin was numbed after 10 minutes immersion.

Two studies were carried out to investigate the influence of habitation to immersion in cold water. The main findings were that regular immersion quickly induced a reduction in shivering at rest. That was accompanied by an equally rapid improvement in isometric endurance There was some evidence of a reduced oxygen consumption at low rates of rhythmic exercise but as the exercise called for a $\dot{V}O_2$ in excess of 1.5 $L.min^{-1}$, there was no evidence that habituation made the exercise more efficient.

# CARDIOVASCULAR AND RESPIRATORY RESPONSES TO MUSCULAR ACTIVITY DURING IMMERSION IN WATER AT DIFFERENT TEMPERATURES

FINAL REPORT
(1977-1981)

by

Alexander R. Lind, D. Phil., D. Sc.
(Principal Investigator)
Thomas E. Dahms, Ph.D.
Carole A. Williams, Ph.D.
Jerrold S. Petrofsky, Ph.D.

Supported by
Office of Naval Research
Contract N00014-77-C-0640
Physiology Department
St. Louis University Medical School
1402 S. Grand Blvd.
St. Louis, MO 63104

# INTRODUCTION

The thrust of this research program was to investigate muscular function during both sustained and intermittent isometric contractions as well as during rhythmic exercise carried out in air or during immersion of subjects to the neck in water at temperatures ranging from 15°C to 35°C. In one experiment the subjects were examined when they were completely under water. The limitations placed on muscular function in those varied circumstances may be related to the limits of flexibility of the cardiovascular and respiratory systems, to the temperature of the muscles and to the metabolic demands in aerobic, anaerobic and fatiguing exercise. Such events may place unexpected demands on both cardiovascular and respiratory function. Isometric exercise, either sustained or intermittent, was chosen to simulate carrying weights underwater, while bicycling, with the legs horizontal was chosen to approximate swimming. Studies were carried out to evaluate the changes that occur as a result of habituation to immersion in cold water. In addition, the mechanisms that compete for the local control of the circulation were examined during intermittent isometric contractions leading to muscular fatigue.

1. Ventilatory responses to exercise in water at different temperatures.

This aspect of the project began as an investigation into the possible interaction between acute cold-induced hyperventilation and the hyperventilation which occurs with isometric exercise. Concern was raised regarding the potentially dangerous consequences of combining these stresses. Based upon the interaction and questions raised by these initial experiments, this aspect of the study was enlarged to include the study of the effects of cold water immersion on the ventilatory response to rhythmic exercise and the effects of habituation on the respiratory responses to both types of exercise.

a) Isometric Exercise

These experiments were of two basic types: one where the subject performed the first of a series of isometric contractions at 40% of the maximum voluntary contractions (MVC) 1 minute following immersion (Type I) and the second set where the first contraction began 10 minutes following immersion (Type II). This design was used so that the first contraction in the Type I experiments would coincide with the period of maximal immersion-related hyperventilation. The Type II experiments were used to assess the respiratory effects of isometric exercise in light of a steady state level of increased ventilation (but not necessarily hyperventilation). These experiments were carried out in water bath temperatures of 15°, 20°, and 35°C. The 35°C water was chosen as a control or thermoneutral water temperature.

The hyperventilatory response ($V_E/VO_2$) to the combined stress (near muscular fatigue) was not different between the Type I and Type II exposures at 20°; however at 15 and 35° the Type I exposure resulted in consistently greater hyperventilatory responses. All subjects perceived the 20° stress as being the most severe because there was little of the "cold anesthesia" or numbness as was experienced at 15°C. So after 10 minutes of immersion at 20°C, the resting level of hyperventilation was increased as was the end exercise level over that of the similar contraction ($C_1$) after 1 minute of immersion in the Type I experiments.

The effect of the water temperature on the hyperventilatory response to isometric exercise was the most striking effect seen in these experiments. Regardless of the type of protocol or the sequence of the contraction, the level of hyperventilation fell nearly linearly with decreasing water teperature (this was the converse of the expected finding). For example, the $V_E/VO_2$ value for $C_1$ in the Type II experiments fell from a value of 122 liters/liter at 35°C to 64 liters/liter at 15°C. This value declined also for the fourth contraction ($C_4$) in this protocol from 113 liters/liter at 35°C to 51 liter/liters at 15°C. Similar changes were seen in the Type II experiments.

The results can only be explained by the inhibition of the neural traffic originating either in the exercising arm or in "central command" by the neural traffic originating from dermal temperature receptors. Such phenomona are known for suppression of pain but the suppression of such a vital drive as ventilation seems unusual but at the same time protective. If these stimuli had been additive the level of alkalosis would have been increased to a serious level. In fact, the end tidal $PCO_2$ at end exercise ($C_1$, Type II) was 10 Torr at 35° but was higher, at 20 Torr, in water at 15°C.

The oxygen consumption increased at the end of the isometric contractions by a greater amount in water at 35°C than in water at 15°C and accounted for most of the decrease in the hyperventilation computation. The minute ventilation response remained unchanged at 20° but fell significantly at 15°C. This effect must have been due to the immersion of the body in the water because it was not seen in the immersion of the forearm only in these water temperatures.

These unexplained results led to the next series of experiments on rhythmic exercise to determine whether this was a reproducible phenomenon in all types of exercise. The question was: Does cold immersion reduce the non-metabolically induced hyperventilation produced with exercise?

b) Rhythmic Exercise:

The influence of cold water immersion on the ventilatory response to rhythmic exercise was tested in both leg and arm cranking of a bicycle ergometer. This ergometer was designed with a double sprocket so that the pedaling occurred below water but the resistance flywheel sat above the bath out of the water. For the arm cranking the pedals were replaced by grips for the hands. The leg exercise experiments consisted of a 10 minute rest period in the water followed by five minute work bouts (continuous at 0,1,2, and 3 Kp at 50 rpm.) This protocol was carried out in 15,20,25 and 35°C water and at room temperature in air at 24°C. The arm exercise was carried out at 0, 1/2, 1 and 1 1/2 Kp at the same temperatures.

The results of these experiments essentially substantiate the finding of the isometric experiment although the ventilatory equivalents ($V_E/VO_2$) were not so high as were found in isometric contractions. The $V_E/VO_2$ relationship is reduced by immersion in cold water.

This effect is seen at rest as well as at lower levels of exercise but not at the highest levels of exercise. These results are generally true for both arm and leg exercise as shown in the following table of $V_E/VO_2$ values:

Table 1: Ventilatory equivalents ($V_E/VO_2$ at rest and during exercise in water at different temperatures, and in air

| | Water Temperature | | | | |
|---|---|---|---|---|---|
| | 35° | 25° | 20° | 15° | Air (24°C) |
| leg exercise | | | | | |
| Rest | 27.3 | 27.3 | 24.4 | 22.1 | 23.7 |
| 0 Kp | 24.7 | 23.7 | 21.2 | 18.6 | 20.3 |
| 1 Kp | 20.8 | 21.7 | 20.3 | 18.8 | 20.8 |
| 3 Kp | 25.8 | 28.8 | 26.5 | 25.9 | 29.9 |
| arm exercise | | | | | |
| Rest | 28.7 | 27.6 | 26.1 | 24.7 | 25.8 |
| 0 Kp | 27.1 | 24.8 | 23.4 | 21.6 | 21.5 |
| 1/2 Kp | 25.5 | 21.5 | 19.3 | 20.1 | 20.6 |
| 1 1/2 Kp | 26.4 | 28.8 | 27.3 | 30.4 | 26.0 |

The general trend does not hold at 15°C water but these ratios are still below those found in air or 35° water. The absence of this effect at 3 Kp (900 Kpm) is undoubtedly the result of this load being over the anaerobic threshold for these subjects which result in a stronger hydrogen-ion induced drive for ventilation. This leads to the conclusion that the ventilatory drive during rhythmic exercise below the anaerobic threshold is of such a nature as to be influenced by whole body immersion or stimulation of large numbers of cutaneous or other thermal receptors.

An additional finding of these studies was the increase in net oxygen cost of the exercise in colder water. That is, the resting metabolic rate increased as did the metabolic rate at each work load and when the net $VO_2$ is computed, it is higher as well. For example, 2 Kp leg work at water temperatures of 35° yielded $VO_2$ values of 1.70 l/min, while in water at 25°C, 20°C and 15°C it was 1.83, 2.13 and 2.00 l/min respectively (see also Table 2 below). This 20-25% increase in oxygen cost of doing work even in proportion at the 0 Kp work load where the increase in $VO_2$ was 120% greater at 15° than at 35° water. The reasons for this highly significant decrease in efficiency are unknown.

In summary, we have seen a significant influence of head-out immersion in cold water on exercise induced ventilatory drive. Unexpectedly, this influence was one of inhibition not facilitation.

2. Cardiovascular responses to isometric and rhythmic exercise in water at different temperatures.

a) Isometric contractions:

The blood pressure response to isometric contractions was unchanged in these experiments, irrespective of the water temperature. The question was pursued further in experiments in which only the forearm was immersed in water for 30 minutes before a contraction at 40% MVC. It was found that the blood pressure response to the contraction was not affected by the water temperature over a range of 15-40°C. The experiments involving immersion of the forearm only were extended to lower water temperatures, between 2°C and 10°C with a 30 minute immersion. In these circumstances there was a small reduction in the pressor response; the mechanism is not known but may be related to temperature-related changes in metabolism which may affect the chemical-reflex component of the pressor response. The possibility that receptors involved in the pressor response are affected at muscle temperatures below 20°C cannot be overlooked; when the forearm was measured in water at 2 and 10°C, muscle temperature had fallen to 20°C or lower during the 30 min immersion of the arm before the contraction. However, in the experiments involving immersion up to the neck, the lowest water temperature was 15°C.

Surprisingly, the reduction in deep muscle temperature in the forearm was exactly the same in cold water during arm-only immerson as it was during whole body immersion where we had anticipated a more rapid fall in the peripheral tissues as part of thermoregulatory defence during whole body immersion.

After 10 minutes of whole body immersion, the resting heart rate for these subjects was not significantly different in water of different temperatures. Further, the increment in heart rate was not different in

response to the isometric contraction during whole-body immersion, irrespective of the water temperature. Similarly, during the experiments when only the forearm was immersed in water, the heart rate at the start and the end of the contraction was not affected by the water temperature.

b) Rhythmic exercise:

The cardiovascular responses to rhythmic exercise exhibited some marked changes when the subjects were immersed to the neck in water at temperatures ranging from 15°C to 35°C. Some of the average data are summarized in Table 2, where the water temperature was 15°C, 20°C and 35°C; data are also given for the same experimental conditions when there was no water in the bath.

Table 2. Cardiovascular responses to rhythmic exercise in air and while immersed in water at different temperatures

| | 15°C | 20°C | 35°C | Air (24°C) |
|---|---|---|---|---|
| Cardiac output $L.min^{-1}$ | | | | |
| Rest | 6.85 | 6.67 | 6.39 | 5.84 |
| 0 Kp | 10.64 | 9.91 | 9.23 | 8.60 |
| 1 Kp | 12.81 | 11.89 | 12.31 | 12.32 |
| 2 Kp | 15.00 | 14.24 | 17.01 | 14.83 |
| 3 Kp | 15.40 | 17.11 | 20.13 | 18.74 |
| Heart rate, $beats.min^{-1}$ | | | | |
| Rest | 71 | 69 | 70 | 66 |
| 0 Kp | 98 | 91 | 91 | 92 |
| 1 Kp | 103 | 102 | 110 | 110 |
| 2 Kp | 116 | 119 | 136 | 132 |
| 3 Kp | 136 | 140 | 159 | 157 |
| Blood Pressure, Torr | | | | |
| Rest | 83 | 94 | 87 | 83 |
| 0 Kp | 92 | 101 | 89 | 87 |
| 1 Kp | 95 | 102 | 90 | 88 |
| 2 Kp | 97 | 108 | 96 | 99 |
| 3 Kp | 104 | 120 | 107 | 109 |
| $\dot{V}O_2$ $L.O_2.min^{-1}$ | | | | |
| Rest | 0.55 | 0.62 | 0.33 | 0.28 |
| 0 Kp | 1.24 | 1.27 | 0.64 | 0.61 |
| 1 Kp | 1.58 | 1.63 | 1.10 | 0.99 |
| 2 Kp | 2.00 | 2.13 | 1.70 | 1.58 |
| 3 Kp | 2.43 | 2.58 | 2.23 | 2.18 |

The relationship between the $VO_2$ and cardiac output was a linear one when the subjects were in air. The slope of the linear regression was y=0.148b-0.665. The data obtained when the subjects were at rest or bicycling in water at 35°C fitted the same linear regression, indicating that the viscosity of the water, at this temperature at least, had no influence on the relationship between the $VO_2$ and the cardiac output. At the two most severe levels of bicycling, the $VO_2$ was higher and was associated with a higher cardiac ouput in water at 35°C than in air.

In water at 20°C, the relationship between $VO_2$ and the cardiac output was also linear (y=0.189b-0.620) but the slope of the regression was substantially steeper than in water at 35°C (or air). The difference was substantial: for example, for a $VO_2$ of 1.0 $L.min^{-1}$ the cardiac output was about 5 0 $L.min^{-1}$ less in water at 20°C than it was at 35°C. Contributing factors to these differences would be the reduction in skin blood flow in the cooler water, and the probability that shivering might have increased the blood flow to inactive muscles. Those two factors would oppose each other in their effect. We have no measurements to assess the influence of either factor. It ought be kept in mind that in cold water some influence of "water-drag" due to the increased viscosity of the water may play a part, as may an increased viscosity of the tissues in the exercising limbs.

While we have no direct way to evaluate the influence of those various possibilities, the data obtained in water at 15°C may, by inference, shed some light on the matter. Here the relationship between $VO_2$ and cardiac output was also linear, at least up to a $VO_2$ of 2 $L.min^{-1}$. The slope of the regression at 15°C was not significantly different from that in water at 20°C although 4 of the 5 points showed a higher cardiac output for the given $VO_2$ values. Those data suggest that a cold dilation of skin vessels occurred in water at 15°C and that any effect of shivering or of increased viscosity of either the water or limb tissues may not be large. Finally, when the work load was increased from 2 Kp to 3 Kp, the increment in $VO_2$ was similar (at about 0.45 $L.min^{-1}$) in water at 15°C as in the other water temperatures, but the cardiac output showed only a small increase. The reasons for a restriction of the cardiac output are obscure. Consideration of all the data on cardiovascular and respiratory responses directs our attention towards peripheral neural imput as one possibly that must be considered.

Much of the difference in cardiac output noted above was due to changes in heart rate; the stroke volume at rest or at given levels of exercise showed only small and irregular differences in water at the various temperatures, a fact that is not greatly surprising since immersion is known to increase the central blood volume markedly.

When the total peripheral resistance was calculated, the values in air and in water at 35°C were similar at rest and at the lowest level of $VO_2$ but diverged as the $VO_2$ reached 1 $L.O_2.min^{-1}$ and higher; the lower TPR at a given $VO_2$ was found in water at 35°C presumably indicating the difficulty our subjects experienced, in subjective terms, of maintaining thermoregulation as the $VO_2$ increased. The TPR for any given level of $VO_2$ was higher in water at 20°C than in water at 15°C, reinforcing the belief that cold vasodilation and shivering occurred in these subjects at 15°C.

Some of the findings are summarized in Table 3, which shows the $VO_2$ at rest, during rhythmic exercise with belt tensions of 0 Kp and 1 Kp during immersion in water at 15°C and 20°C on the first and last days of the habituation experiments. For comparison, values are also shown for water at 35°C and for air at 24°C.

Table 3. Effect of habituation on oxygen uptake at rest and while bicycling.

| | | WATER TEMPERATURE | | | |
|---|---|---|---|---|---|
| | | 15°C | 20°C | 35°C | Air (24°C) |
| Rest | First Immersion | 0.68 | 0.56 | 0.33 | 0.33 |
| | Last Immersion | 0.50 | 0.36 | | |
| 0 Kp | First Immersion | 1.28 | 1.25 | 0.68 | 0.58 |
| | Last Immersion | 1.23 | 1.10 | | |
| 1 Kp | First Immersion | 1.69 | 1.63 | 1.09 | 1.16 |
| | Last Immersion | 1.71 | 1.58 | | |

The values in water at 35°C are not different from those found in air, suggesting that water drag (at that temperature) was not a large factor. Of course, the increasing viscosity of water as its temperature decreases may play a part in the increased $VO_2$.

The results at rest show that habituation resulted in a fall in $VO_2$ (associated with visual evidence of a reduction of shivering). During exercise at 0 Kp, there was a reduction in $VO_2$ with habituation in water at 20°C but not in water at 15°C. When the bicycling was at 1 Kp the habituation had no effect on the $VO_2$ in either water temperature. The values for $VO_2$ during exercise in the cold water were much higher than in water at 35°C or in air. It is not clear what the causes are for those differences but during exercise presumably the increased viscosity of the water and of the tissue in the leg play a part, while the increased heat loss by convection during exercise may well have prompted shivering to occur.

4. Investigation of the diving reflex:

Experiments were conducted to determine whether the "diving reflex" is exhibited by man upon total body immersion in water. Classically, mammals demonstrate a bardycardia and peripheral vasoconstriction during a dive. This is accomplished by a combination of two reflexes: one is neurally mediated and the other is chemically mediated. In the first, the sensory endings of the trigeminal nerve are activated when the face is immersed. Activation of these endings cause a reflex apnea, a vagally-induced bardycardia and sympathetically-induced reduction in blood flow to skeletal muscle, splanchnic and renal vascular beds. Cutaneous veins are also constricted. The second reflex comes into play as a result of the apnea, which causes a reduction in $PCO_2$ in the arterial blood. These changes activate arterial chemoreceptors, which in turn cause circulatory changes that enhance those induced by the activation of the trigeminal nerve, namely, further reduction in the heart rate and peripheral vasoconstriction. As long as the face is immersed and there is continual sensory input from the trigeminal nerve, any modification of the respiratory centers from the chemoreceptors are overriden.

This complex series of events represent the mechanisms responsible for maintaining the arterial blood pressure and therefore adequate oxygenation to the two most vital regions, the brain and the heart.

Four weeks after the control cold immerson at 20°C, the subjects were exposed to the described procedure for four days in water at 15°C, and on the fifth consecutive day, again in water at 20°C for comparison with the control

experiments. The habituation procedure was not prolonged further because of our earlier experience that the greatest effect of habituation occurs early in serial exposures (see above).

The main thrust of these experiments was to assess the $VO_2$ at rest and during pedalling at 2 levels of severity. Actually, two sets of controls exist, first between the first and final immersion in water at 20°C and between the first and fourth (final) immersion at 15°C (which interposed between the two immersion experiments in water at 20°C).

At rest: The first immersion in water at 20°C resulted in a $VO_2$ in minutes 8-9 (before the start of exercise) of 0.56 $L.O_2.min^{-1}$ a 39% increase over the $VO_2$ at rest in air at 24°C. After the habituation procedure the $VO_2$ at rest in water at 20°C had fallen to 0.37 $L.O_2.min^{-1}$ (a reduction of 34%) a value which was not significantly different from the $VO_2$ in air.

In the first of four days immersion in water at 15°C, the $VO_2$ at rest was 0.67 $L.O_2.min^{-1}$ or 50% greater than the value in air at 24°C. On the fourth day of immersion in water at 15°C the resting $VO_2$ had reduced by 24% to 0.51 $L.O_2.min^{-1}$ which was, however, still 35% greater than that found in air.

Pedalling at 0 kp belt tension: On first immersion in water at 20°C, rhythmic exercise at this level resulted in a $VO_2$ of 1.25 $L.O_2.min^{-1}$, more than double the value for the same external work in air which was 0.57 $L.O_2.min^{-1}$ ($\Delta$ 0.68 $L.O_2.min^{-1}$). On the last day of habituation, the energy cost of this level of work was reduced by 10% to 1.10 $L.O_2.min^{-1}$.

Over the four days of immersion in water at 15°C, however, there was no significant change in the $VO_2$. The actual values were 1.33 $L.O_2.min^{-1}$ on day 1 to 1.24 $L.O_2.min^{-1}$ on day 4. Clearly, both values were numerically higher than those for the first and last days of immersion at 20°C.

Pedalling at 1 kp belt tension: On the first day of immersion in water at 20°C, the $VO_2$ was 1.63 $L.O_2.min^{-1}$, compared to 1.16 $L.O_2.min^{-1}$ in air ($\Delta$ = 0.47 $L.O_2.min^{-1}$). After the habituation exposures, there was no significant decrease in the $VO_2$ in water at 20°C; the average value was 1.58 $L.O_2.min^{-1}$. Similarly, there was no difference between the energy cost of bicycling at this level on the first and last day of immersion in water at 15°C, when the average values of $VO_2$ were 1.69 and 1.71 $L.O_2.min^{-1}$, respectively. There was no significant difference in the values for $VO_2$ in water at 20°C or 15°C though the numerically higher values in the colder water indicates that the matter is one of degree and part of a continuum of increased $VO_2$ as the water temperature decreases. That is evident in other experiments (see above) when the findings at rest and during exercise are compared to those when the subjects were immersed in water at 35°C, which were not different from those found in air.

reduced enough to expect a reduction in the MVC and that was, in fact, confirmed here. The blood pressure response to the isometric contraction was unchanged with habituation.

Measurement of the gasp response (the time to lowest $PCO_2$) showed modest increases in time (varying from 1 to 23 sec) after immersion, while the time taken to return of $PCO_2$ to control values about halved. But the time to the gasp response never exceeded 23 sec and the recovery was also complete within 3 min. It is not easy to see how respiratory exchanges were important to the endurance times of isometric hand-grip contractions after 10 min immersion.

From other experiments (see below) there was little difference in the forearm blood flow during hand-grip contractions during immerson in water at 15-35°C so that it is not likely that there is a serious functional deprivation of blood flow to the active muscles in water at 15°C.

The remaining likely possibility to explain the improved isometric endurance with habituation seems to be that shivering induces some level of muscular fatigue so that on the first experience of immersion in cold water, there is a considerable encroachment on the isometric endurance. With habituation and the consequent reduction in shivering, the isometric endurance would then be expected to increase. This hypothesis would be hard to test experimentally without invasive procedures to assess the components of the venous effluent from the muscles under study or by standardized electrical stimulation,procedures that are not considered to be without risk in such experiments.

From this study it is clear that habituation to immersion in cold water results in a marked improvement of isometric endurance in limb muscles and a marked reduction in shivering. The subjects also reported a considerable reduction in subjective discomfort during the period of habituation. The major improvements occur early in a series of days of immerson but the improvement continues albiet at a slower pace, over a period of 2 weeks. During this experiment, there was some evidence to suggest that some of the effects of habituation was lost over the weekend between the two bouts of 5 consecutive days of exposure; however, our data show no statistically different changes over those two days without immersion. Overall, the changes we have found fit the general picture of acclimation to cold air seen between, for example, Australian aborigines and non-acclimated Caucasian subjects exposed to cold nights in the desert. Shivering is generally regarded as an uneconomic and a distinctly uncomfortable way for the body to try to maintain its core temperature.

b) Rhythmic Exercise: The second experiment was concerned with the changes brought about by habitual immersion in cold water both at rest and during rhythmic exercise at two different levels of severity. Three subjects (of whom one had taken part in the previous year's habituation experiments) were immersed to the neck in water for 10 min at rest, followed by 5 min pedalling on a Monark bicycle ergometer at 0 Kp belt tension and immediately after by a further 5 min pedalling at 1 Kp belt tension. The bicycle ergometer was suspended above the water on a frame with chains to the pedals which were in the water at hip level, so that the seated subject worked with the legs close to horizontal; this procedure ensured that the belt was not wetted and did not alter in tension. The control experiment in water at 20°C, was part of another series of experiments in which the subject pedalled at belt tensions varying from 0 Kp to 3 Kp while in air or immersed in water ranging in temperatures from 20-35C.

The data on changes in blood pressure are equally interesting. In all experimental conditions, the highest blood pressure either at rest or during rhythmic exercise occurred in water at 20°C, some 7-15 mmHg higher than in water at 15°C, 35°C or in air. The values in water at 15°C and 35°C were similar. On a sensory basis, the subjects were unanimous that they were most uncomfortable in water at 20°C and that in water at 15°C they felt numb after 10 minutes immersion.

In practical terms, it is clear that undesirably high energy costs occur when rhythmic exercise is performed in water at 20°C and, particularly at lower water temperatures, not only is the energy cost increased, a limitation seems to be placed on the ability of the cardiovascular system to respond, leading to an encroachment on the cardiac reserve.

3. The effects of habituation to immersion in cold water on muscular performance:

Two experiments were carried out.

a) Isometric Contractions: The resting energy expenditure of the subjects immersed in water at 15°C decreased by 25% as a result of habituation. While we have no other objective measurement of the shivering process, it was obvious from visual inspection that the degree of shivering was the greatest on the first immersion, and was reduced in all subsequent immersions. The marked reduction in $VO_2$ and the visual observations were supported by the subjects' comments which made it clear that they experienced much less discomfort with successive immersions.

The reduction in $VO_2$ was not evident in the changes in rectal temperature with successive immersions. The rectal temperature consistently fell by 0.4°C in 10 minutes of immersion in water at 15°C. While that was not particularly surprising, keeping in mind the slow response of rectal temperature to changes of heat load, whether endogenous or exogenous, it was surprising to find no change in rate of cooling in the forearm muscles when the whole body was immersed when compared to immersion of only the forearm. The temperature of the muscles, measured at a depth of 15 mm, was remarkably consistent in those circumstances. It was measured during the first (control) exposure and again on the first and tenth days of the habituation experiment. The average for muscle temperature was 34.4°C before immersion, 34.2°C after one minute of immersion and 29.2°C after 10 minutes of immersion, just before the first isometric contraction.

The endurance times of the isometric contractions were also puzzling. One of the most profound influences on isometric endurance is muscle temperature; the longest contractions are associated with a muscle temperature in the range of 28-30°C, above and below which there is a marked reduction in endurance time. In the present study, the endurance time was increased by nearly 30% ($P<0.05$) from the first (control) immersion to the first immersion of the habituation study. Thereafter, the endurance time increased numerically by 9% on the tenth day of the habituation study but that apparent increase failed to reach statistical significance ($P<0.10>0.05$). Since the muscle temperatures were identical in all immersions, some factor other than muscle temperature, must have influenced the endurance times. The skin temperature is presumed to have been similar in all these immersions. The changes in endurance time cannot be readily explained by the measurements made. The only major difference noted was in the $VO_2$ as the shivering decreased from the first (control) immersion and through the subsequent habituation immersions. The muscle temperature was not

Based upon this well-documented series of events, head-in immersion should cause a bradycardia and a reduction in the forearm blood flow when compared to head-out immersion. There should also be an augmented vasoconstrictor effect seen on the forearm blood flow if the immersion occurs in a water temperature colder than the "normal" arm and skin temperature.

The forearm blood flows were measured by venous occlusion plethysmography from subjects who were first immersed up to the neck, with the head out of the water at temperatures of 15, 20 and 35°C. In these experiments, the test arm was kept at heart level. Isometric contractions of the forearm muscles were exerted at 10% of the subject's maximum voluntary contractin (MVC) using a hand-grip dynamometer.

The changes in forearm blood flow were measured at rest and during the isometric contraction at 10% MVC when the subjects were immersed in water to the neck and also to whole body immersion (i.e., head under water)are summarized in Table 4. Following the "head out" immersion during which a contraction was exerted at 10% MVC the subject submerged himself and repeated the exercise in water at 20 or 35°C. While under water the subjects breathed through a low

Table 4: Forearm blood flows at rest and during a hand-grip contraction at 10% MVC in water at 20°C and 35°C with immersion to the neck and whole body immersion (head under).

Forearm Blood Flows ($ml.min^{-1}.100\ ml$)

| | "Head out" | | "Head in" | |
|---|---|---|---|---|
| Water Temperature | Rest | 10% MVC | Rest | 10% MVC |
| 20°C | 1.8 | 4.9 | 2.5 | 6.3 |
| 35°C | 4.0 | 6.0 | 3.8 | 6.2 |

resistance mouthpiece. The resting blood flows were lower in water at 20°C than in water at 35°C, though the values at each water temperature were not significantly different whether the head was out of the water or under it. The blood flow increased during the isometric contraction to levels that were not significantly different regardless of the water temperature; this was the case for both the "head out" and "head in" experiments.

The most plausible explanation for these findings appears to be that the subjects were allowed to breathe freely through a low-resistnce mouthpiece during whole body immersion. This eliminated a large portion of the signal responsible for the circulatory changes expected during immersion, and probably accounts for the lack of any demonstrable vasoconstriction in the forearm. It points out that the respiratory reflex control is dominant in the "diving reflex" over the reflex generated from the trigeminal area.

5. Control of the local circulation during intermittent isometric contractions.

Earlier studies had shown that the blood flow through a forearm performing intermittent isometric contractions at 60% MVC (4 sec contractions with 8 sec intervals) resulted in muscular fatigue, associated with a large increase in perfusion pressure. But the blood flow to the limb between those contractions was constant, and only about half the maximal flow possible. In the face of a steadily increasing blood pressure, there must have been local vasoconstriction. A series of experiments gave results which indicated that in the normal human forearm the myogenic reaction of blood vessels could not be held responsible for these findings. That left, as the remaining explanation, the possibility that there was a neurogenic vasoconstriction. Experiments were devised to investigate the problem. Control experiments on male and female subjects were followed by close-arterial infusions of an $\alpha$-blocking agent, a $\beta$-blocking agent, both those agents combined and of the $\alpha$-blocking agent in the presence of its antagonistic drug. The results make it clear that the failure of the local blood flow to increase in sequential intermittent contractions and in the face of a marked increase in perfusion pressure is indeed due to a sympathetic adrenergic constriction mediated by $\alpha$-receptors. The administration of phentolamine resulted in a sharp increase in blood flow which, during the experiment, increased with the rising blood pressure, with no change in forearm vascular resistance. In experiments following administration of propranolol with phenylephrine preceding the phentolamine, the results were not different from the control condition.

In another study, the suggestions made in recent years that during sustained isometric contractions, the blood flow in a contralateral arm inevitably increased, mediated principally by $\beta$-receptor sympathetic activity, were examined. We have found, in agreement with earlier conclusions, that there is no increase in the blood flow through the contralateral arm except in the presence of electromyographic activity. Neural complicity in this control has also been shown to be due to the $\alpha$-adrenergic receptors.

In animals, we have examined the changes in blood pressure induced by isometric exercise induced artificially by electrical stimulation which "mimics" voluntary contractions. Importantly, we have found that isometric contractions of the soleus muscle (all slow-twitch) fibers) in the cat elicit little or no increase in arterial blood pressure. When muscles with a mixed population of fiber types, such as the gastrocnemius, were activated, there was a marked increase of blood pressure, which was little affected by "selective" blockade of slow-twitch fibers (curare) but was substantially reduced when fast-twitch fibers were "selectively" blocked (hexamethonium). The inference is that slow-twitch mtor units contribute much less than do the fast-twitch fibers to the increased blood pressure in isometric contractions. The question of the mechanisms responsible and the practical implications of the afferent neural pathways are of great importance and remain to be elucidated.

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